Novel Treatise Concerning Intestinal Microbiome Dynamics - Induction of Enhanced Mutagenicity of Good Bacteria as Path Forward

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Introduction

Current doctrine holds that the resolution of certain intestinal ailments associated with the prevalence of "bad bacteria," particularly strains such a C. Difficile, can be remedied by introducing "good bacteria." While some reports indicate that the introduction of good bacteria can ameliorate mild to moderate diarrhea associated with oral-route antibiotics, this is true only when live and active cultures of comparatively high density are ingested in synchrony with each dose of oral route antibiotic. With conditions such as C. Diff. infection, probiotics, in fact, have a exacerbating effect on symptoms as they also do when antibiotic-associated diarrhea has already set in prior to culture ingestion.

Fecal Matter Transplant (FMT) has been demonstrated, in many cases, to be effective in eradicating C. Difficile infection but the underlying mechanism of the efficacy of this therapy has never been properly attributed. In some cases, this therapy does not succeed in resolving the infection.

There are clearly fundamental gaps in the current medical understanding of the dynamics between so-called "good" and "bad" bacteria, particularly considering that probiotics exacerbate diarrhea for many. These side-effects often go unreported because probiotics are not regulated in the same way as prescription drugs and doctors have no mechanism for reporting feedback from patients concerning the efficacy of home remedies. New theory is required in order to fill in these gaps in our understanding so that potentially harmful medical advice may be replaced with more appropriate advice in the future.

Abstract

Even in medical literature, the dynamical relationship between different strains of bacteria in the colon is often referred to in terms of one species/strain "crowding out" the other as if they were competing for common food sources. This description is extremely misleading and amongst those who have been mislead are our own researchers.

Firstly, the prevention of diarrhea before it has begun in the case of probiotic ingestion alongside oral-route antibiotics may have more to do with the absorption of penicillin enzymes in the small intestine and appendix, preventing large quantities of unabsorbed penicillin from entering the colon and creating the problem in the first place. Once antibiotic-associated diarrhea has set in, however, the addition of probiotics exacerbates symptoms for reason that the

"good" bacteria themselves are introducing a food source which is palatable to the "bad" bacteria.

Strains of bacteria considered to be 'bad' are 'bad' insofar as they tend to replicate as much as possible and release inflammatory compounds, resulting in the inflammation of the colon. Under normal conditions, these bacteria do not spread out of control for reason that the diversity of good bacteria, which enjoy greater mutagenicity the greater the diversity of the species present in the environment, undergo regular changes to their protein signatures, which can be "sniffed" by the bad bacteria. *If the good varieties of bacteria do not mutate over time at a sufficient pace, the bad bacteria become accustomed to the signaling proteins of these strains and begin to identify them as being of the same species and thus, a possible energy source.* Under ordinary circumstances, bad bacteria borrow chemical energy in the form of sugars from the interior of neighboring bacteria of the same species, exclusively, but not from other species. Extant research, however, tells us that bacteria generally are capable of incorporating DNA fragments from other species into their own DNA, driving the mutation process. When bacteria coexist in an environment with many other strains, this maximizes the rate of mutation. The "bad bacteria"'s theft of nutrients, provided it is restricted only to its own species, enables new cellular genesis only at the expense of effectively starving other cells, keeping growth to a minimum and producing the splotchy migration of bacteria familiar to biologists in which a space occupied by a colony becomes vacant before long and in which unoccupied spaces become occupied.

It is not the quantity of good bacteria which is relevant to the dynamic of good versus bad bacteria, but rather, the rate of mutagenicity. When good bacteria are introduced in large quantities in an attempt to alleviate the symptom of diarrhea, the bad bacteria are actually being provided with a palatable food source for reason that commercially available probiotics are bred from common baseline examples of good bacteria rather than new varieties. Even if commercially available probiotics were varied infinitely, they would not eradicate extant good bacteria which act as a persistent food source for the bad bacteria. What is needed is not new bacteria or even different bacteria, but chemical factors which drive mutation of bacteria.

The bad species of bacteria use nano-tubules to penetrate the membranes of good bacteria as well as bacteria of their own species in order to extract sugars. Unlike a parasite, these bacteria are very particular about from whence they obtain chemical energy and will not attempt to extract chemical energy from bacteria which appear unfamiliar. After hundreds of reproductive cycles in the presence of a particular array of good bacteria species, bad bacteria may begin to replicate out of control as a consequence of their newfound appetite for sapping these cells of their chemical energy.

In the case of FMT, the combination of irrigation of the colon to remove much of the C. Diff. infection combined with the complete removal of good bacteria combined with the introduction of a wholly unique set of colonies of good

bacteria from a healthy donor result in the denial of palatable food sources for any remaining C. Diff colonies.

In those cases in which FMT is not effective, it is almost certainly due to an incomplete flushing of good bacteria from the colon prior to the introduction of the donor FM. The old "good bacteria" may continue to act as a food source which revives the latent C. Diff and enables a new foothold by the strain whereas the complete elimination of all good bacteria varieties known to the bad bacteria results in those harmful strains eschewing potential food sources even in the face of starvation.

Conclusion

Armed with this new understanding, new guidelines can be established including recommendations for *variable acidity* diets which promote mutations of established good bacteria (as mutations are a natural response to highly variable environmental conditions) and which could be predicted to enhance the variety of good bacteria present in the intestine over time. Literally consuming bacteria is not advisable for those affected but rather eating a varietal diet in which macronutrient groups are not co-mingled (also effective for ameliorating diabetes, incidentally) and ensuring the same meals are not consumed repetitively.

It is also worth noting that hypotheses which have suggested that the microbiome is linked to weight loss or weight gain are specious. In many cases, recipients of FMT who experienced the resolution of C. Diff. infection experienced obesity after receiving the transplant, but these reports do not account for the obvious fact that C. Diff sufferers are known for over-eating to compensate for diarrhea-associated weight loss and often continue in this habit after resolving their C. Diff infections out of habit, resulting in the weight gain.

In summation, "bad bacteria" can be most accurately characterized as 'bad' by their induction of inflammatory effects combined with their ability to rob surrounding bacteria of nutrients; a behavior not exhibited by all species of bacteria but one which this author believes depends upon the perceived familiarity of those bacteria by the harmful colonies.